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Inside This Issue

The Science and Art of EMG Testing

Eric J. Sorenson, MD

"Much of neurology hinges on locating the problem. Once you've established the location, you have a much greater likelihood of determining the cause and fixing it. One of the great strengths of EMG [electromyography] is its high level of accuracy in localization." With these words, Eric J. Sorenson, MD, helps explain the importance of EMG in diagnosing and treating neuromuscular disease. Director of the EMG Laboratory in the Department of Neurology at Mayo Clinic Rochester, Dr Sorenson notes that EMG can determine which nerves and muscles are impaired and whether the source is the nerve, the neuromuscular junction, the muscle, the spinal cord, or a combination of structures.

Anyone who has listened to the buzz and crackle of an EMG recording knows that interpreting EMG responses requires fine auditory discrimination and an experienced ear. EMG is not an exact science—neither the test protocols nor the results are black-and-white (Figure). EMG must be conducted and interpreted within the context of the clinical examination and clinical hypotheses. C. Michel Harper, MD, a neurologist and former director of the EMG Laboratory, points out that EMG "is not an end in itself, but rather an extension of the neurologic examination, much as a tuning fork or tendon hammer. It is a tool that allows physicians to probe specific aspects of the nervous system."

Context and Bias—Sorting the Wheat From the Chaff

EMG is always subject to bias, and in the hands of an experienced electromyographer, bias is helpful and important. Dr Sorenson explains: "Because EMG is so sensitive, it's not uncommon to have false-positive results, so it is critical to distinguish true- and false-positives, to sort the wheat from the chaff." Dr Harper adds: "Narrowing down the pretest probabilities allows us to collect less EMG data to answer the diagnostic question, to recognize incidental findings that have nothing to do with the history or clinical exam findings, and to consider whether an extraneous finding is meaningful or not."

C. Michel Harper, MD

The more skilled one is at EMG,

- the fewer recordings are needed to answer the diagnostic question.
- the easier it is to attend simultaneously to the electrical response and patient comfort.
- the better one is at distinguishing relevant from irrelevant information and outlier responses.

A large patient volume and varied caseload that includes complex cases such as brachial plexus injuries and rare peripheral neuropathies enhance EMG expertise and specialized skills. Mayo Clinic electromyographers, for example, routinely perform single-fiber EMG, a technique that samples 1 or 2 muscle fibers rather than the standard 10 to 15. It is difficult to perform, not widely available, and 95% to 100% sensitive in diagnosing myasthenia gravis.

Because EMG is not an exact science, training and mentorship are particularly critical. Residents at Mayo Clinic's 3 sites train for at least 6 months in the EMG lab—this is 3 months longer than most other residencies. Many of their mentors trained under Edward Lambert, MD— one of the forefathers of EMG who established the nation's first EMG lab at Mayo Clinic in the 1950s—and also with Jasper R. Daube, MD, Andrew G. Engel, MD, Donald W. Mulder, MD, Peter J. Dyck, MD, and others whose contributions to the understanding of neuromuscular disease continue to inform refinements in the technique, science, and practice of EMG.

EMG at Mayo Clinic: One Lab—Integrating Patient Care Across Disciplines and Sites

Mayo Clinic Rochester

- has one of the largest EMG labs in the world.
- conducts more than 15,000 EMG studies a year.
- conducts monitoring in approximately 500 surgical cases a year.

At Mayo Clinic Rochester, 11 neurologists and 4 physiatrists work in the EMG lab and operating room on a rotational basis. Approximately 25 allied health staff and technicians, many with more than 20 years' experience, work in the lab and on surgical cases. While most institutions have separate EMG labs for neurology and physical medicine, Mayo's lab serves both disciplines in patient care and resident training. Dr Sorenson says, "Mayo has a very collegial environment. Everybody shares and does what they do best.

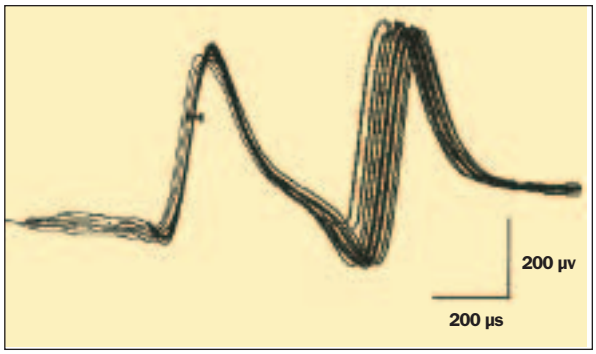

Figure. Two muscle fiber action potentials recorded during single-fiber EMG.

Our EMG lab is an example of that type of integration and collaboration." Dr Harper adds, "In most places, patients get a totally different exam, depending on what 'door' they use to enter the lab. At Mayo, all patients go through 1 door, regardless of the nature of the problem."

EMG techniques, lab organization, and philosophy of practice are consistent across Mayo's 3 sites. The staffs at Mayo Rochester, Arizona, and Jacksonville have a collaborative relationship and gather several times a year to discuss clinical practice, share research, and participate in continuing education.

Pediatric EMG: Combining Expertise and Compassion

The advent of "awake anesthesia" or "conscious sedation" has made EMG in children feasible. It allows compassionate testing under moderate sedation. The patient is awake enough for voluntary movement, but sedated enough to minimize discomfort. Mayo Clinic Rochester conducts these studies in a special suite attached to an operating room with a dedicated nurse anesthetist supervised by a pediatric anesthesiologist. As Dr Harper says, "The art of EMG in children is conducting the test in the shortest time with the least amount of discomfort and working in an integrated way with the anesthesiologists who are experts at titrating sedation in an environment that's really safe. Families of children who have had EMGs elsewhere say the experience at Mayo is just absolutely different."

Brachial Plexus Surgery and EMG

Traumatic and extensive brachial plexus neuropathy can leave an arm useless and be difficult to repair. EMG helps to identify and map out impaired nerves before surgery and to determine if nerve grafts or nerve transfers are needed (ie, if a nerve root is functional). As Dr Sorenson says, "These are delicate surgical procedures, and the more the surgeon knows about what he or she

Table. EMG Referral

Common symptoms that trigger referral
Weakness, numbness, sensory loss, neuropathic pain, or a combination of these findings
Common conditions include
Brachial and lumbar plexopathies
Carpal tunnel and cubital tunnel syndrome
Motor neuron disease
Myopathies (congenital and acquired)
Neuromuscular junction disorders (eg, congenital and acquired myasthenia gravis, Lambert-Eaton myasthenic syndrome)
Nerve terminal disorders
Peripheral neuropathy
Radiculopathies
Common referral sources within Mayo Clinic
Neurology (peripheral nerve, neuromuscular, or general)
Child and Adolescent Neurology
Neurosurgery
Spine Center
Internal Medicine
Orthopedic Medicine (Hand Clinic)

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PHYSICAL MEDICINE AND REHABILITATION

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Kathryn A. Stolp, MD
Jeffrey A. Strommen, MD

has to work with before surgery the better." When the nerves are exposed during surgery, they are directly stimulated with nerve action potentials and EMG. "We also stimulate the motor and sensory cortex, testing the entire motor and sensory pathways," explains Brian A. Crum, MD, a neurologist who routinely conducts neurophysiologic monitoring during surgery.

Surgical Monitoring

At Mayo, depending on the type of surgical case, other types of neurophysiologic monitoring such as evoked potentials may be used in addition to or instead of EMG to monitor the integrity of the cranial nerves and motor and sensory pathways. In patients with acoustic neuroma, for example, acoustic or auditory nerve and facial nerve function are monitored. Dr Crum states: "The amount of monitoring at Mayo is more extensive than it is in many clinical facilities because our cases are often complex. We participate in several surgical procedures each day. Our goal in all neurophysiologic surgical monitoring is to prevent unnecessary neurologic damage by doing the safest procedure. We are fortunate to have technicians with 20-plus years of experience."

Whether performing a complicated brachial or lumbar plexus repair, removing a brain, spine, or peripheral nerve tumor, determining the nature and source of muscle weakness of unknown etiology, checking the accuracy of a diagnosis of myasthenia gravis, or managing a routine carpal tunnel procedure, the success of EMG depends on more than state-of-the-art equipment. Prestudy narrowing of the differential diagnosis based on clinical findings, sensitivity to patient comfort, a steady hand, experienced visual and auditory discrimination skills, and efficient data collection in the lab and during surgery can make EMG a valuable extension of the neuromuscular examination and an important tool in surgical repair.

Patient Referrals

Referrals from within the Mayo Health System can be made directly to the EMG lab in Rochester. Referrals from outside the Mayo system generally go through the individual departments (Table).

Because a direct correlation between the neurologic examination findings and EMG study is so critical, patients may be required to have a repeat EMG, regardless of the how recent their previous EMG may have been. Dr Harper notes, "Referring physicians don't have to worry about ordering an EMG ahead of time, because once the patient is at Mayo, the EMG, if it is indicated, will be done within 24 to 48 hours."

Repairing the Nervous System: Remyelination and Multiple Sclerosis

Acute inflammatory demyelination, limited remyelination, progressive axonal loss, and development of multifocal sclerotic plaques—this is the typical sequence of injury to the brain in patients with multiple sclerosis (MS). For patients, this translates into a difficult-to-predict course of sometimes benign but often debilitating inflammatory attacks that are commonly followed by degenerative decline. For physicians, it represents one of the most common reasons for neurologic referral, a major cause of disability in their young adult patients, and a heterogeneous and enigmatic neurologic disease.

Claudia F. Lucchinetti, MD, Moses Rodriguez, MD, and Brian G. Weinshenker, MD

MS occurs when a confluence of poorly characterized genetic and environmental factors initiates an immune-mediated response directed at brain components. T cells break through the blood-brain barrier, mistakenly identify healthy myelin-producing cells or their myelin products as foreign, and attack and destroy them.

Symptomatic treatments are often successful, but no treatment can stop the progression of the disease. As Claudia F. Lucchinetti, MD, a neurologist and MS researcher at Mayo Clinic Rochester, puts it, "We're still at a point where many patients worsen, despite therapies that impact the inflammatory aspect of the disease. So the focus of MS research is understanding why this occurs and what can be done about it."

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Brian G. Weinshenker, MD

Downregulating the Immune System—Positive or Negative?

Paradoxically, the inflammatory immune response that produces demyelination also induces remyelination, a natural repair process of demyelinated tissue. Natural remyelination is spotty, patchy, and limited in the central nervous system (CNS). Yet suppressing inflammation may prevent it altogether.

Twenty years ago, when the idea that the CNS could repair itself was unheard of, Moses Rodriguez, MD, a Mayo Clinic neurologist and researcher, was trying to prove that stimulating the immune system would aggravate an MS-like illness in animals. In fact, the animals' condition improved and they showed evidence of remyelination, indicating CNS repair not only was possible, but also was enhanced by increased inflammation directed against the CNS. Converging lines of evidence, including brain biopsy and autopsy tissue analysis, confirmed that remyelination, as well as demyelination, occurs in humans with MS.

Today, uncovering the mechanisms of successful and unsuccessful remyelination in MS is considered a critical step in preventing disease progression. Myelin sheath destruction results in acute, usually transient and reversible inflammatory attacks that interfere with axonal conduction of neuronal impulses. But failure to remyelinate eventually leads to axonal death and the chronic, irreversible motor, sensory, and cognitive deficits of late-stage MS. It is this chain of events—from acute attack to downstream destruction—that Mayo researchers want to prevent.

Two Phases of MS, Two Patterns of Tissue Injury

The Inflammatory Acute Phase

The early phase of MS is characterized by intense inflammation, focal demyelination, and limited remyelination during acute attacks of neurologic dysfunction. It occurs in the context of T-cell invasion across the blood-brain barrier. Axons may or may not recover during this phase. Symptoms are treated with varying degrees of success by immunosuppressive, anti-inflammatory, or immunomodulatory drugs, alone or in combination. Plasma exchange, first studied in a controlled trial at Mayo Clinic as a possible "rescue therapy" for catastrophic refractory episodes, is effective in some forms of MS.

The Chronic Progressive Phase

There is no treatment for the chronic progressive phase. Not only myelin, but also neurons are injured in the cerebral and cerebellar cortices. Global tissue destruction in chronic MS occurs in the context of a relatively intact blood-brain barrier. Because the process is compartmentalized in this way, it is likely less accessible to the effects of anti-inflammatory or immunomodulatory treatments.

The smoldering inflammation, demyelination, and axonal injury of this phase probably begin during earlier inflammatory attacks. The process affects demyelinated plaques as well as normal-appearing white matter. Wallerian degeneration and loss of trophic support from growth factors associated with healthy oligodendrocyte cells are likely involved. Researchers believe that perturbed interactions between axons and myelin sheaths interferes not only with conduction and remyelination, but also with axonal outgrowth and connectivity. Eventually demyelination disturbs the delicate balance and complex physiology of the entire CNS.

Brian G. Weinshenker, MD, a neurologist and MS researcher at Mayo Clinic Rochester, reports that patients often see the change from the acute to the chronic phase as an abrupt switch. However, even in early MS, MR spectroscopy shows reduction of axonal density. The brain has considerable reserves, but at some point, axonal death reaches a tipping point, and patients begin to experience progressive loss of neurologic function.

The question is, can it be prevented and, if so, how? The answer may lie in therapeutic remyelination.

Remyelination: Keeping Axons Alive

Remyelination keeps axons alive, but natural remyelination is limited. Could the process be stimulated therapeutically? Even without reestablishing conduction, keeping axons alive might limit or prevent the diffuse damage and lasting disability of chronic MS.

Therapeutic remyelination has become a beacon of hope, and the team of MS neuroscientists and neurologists at Mayo Clinic are in the forefront of research to make it a reality.

Patterns of Natural Remyelination

Are some people better at remyelination than others? It turns out they are. The MS Lesion Project, funded by the National Multiple Sclerosis Society and led by Dr Lucchinetti and a diverse international group of collaborating scientists, identified more than 700 people with MS who underwent brain biopsies. From these data, they discovered not only evidence of remyelination, but 4 distinct MS subtypes. Two show evidence of good natural remyelination. Two do not. In a separate study funded by the National Institutes of Health, Dr Lucchinetti and colleagues are investigating whether genetic predisposition contributes to suc-

cessful natural remyelination.

Antibody 22, a Key to Remyelination

Early thinking about therapeutic remyelination focused on transplanting cells such as oligodendrocyte progenitor cells and on administering neurotrophic growth factors. However, there are numerous unresolved issues in cell transplantation, and growth factor strategies have had serious adverse effects and disappointing results.

Dr Rodriguez, Larry R. Pease, PhD, and colleagues were on another track—the search for natural human monoclonal antibodies that would promote repair. Some of these antibodies are involved in the demyelination; some in remyelination. As Dr Rodriguez says, "Each MS lesion is its own little world composed of destructive and protective factors. We tried to enhance the reparative factors."

In 2001, as Dr Lucchinetti's team was uncovering MS subtypes, Dr Rodriguez and his colleagues discovered the natural monoclonal autoantibody they were after. Unlike the antibodies made by industry to fight cancer, it is an immunoglobulin of the M type (an IgM). They labeled it "number 22." Then, using DNA from the patient in whom they discovered it, they sequenced and reproduced it in recombinant form.

Figure 1. A schematic of an IgM. An IgG would consist of just one of the subunits.

Number 22 differs from other antibodies. First, it is an IgM, the largest type of antibody with 5, rather than 2 binding sites (Figure 1). Second, it occurs naturally in many individuals and species and is very primitive in evolutionary terms. It is the body's first and most rapid line of defense, often referred to as the "innate immune response." Because it is natural, it carries few if any adverse effects, unlike industry-synthesized IgGs. Dr Rodriguez and his team found that it promoted remyelination in approximately 50% of lesions in mice with a virus that mimics MS and was effective in more than 85% of animals tested (Figure 2). They immediately set about producing enough to take to human trials. Now, in conjunction with a Good Manufacturing Practices facility at the University of Minnesota, they have made enough to be steps away from a US Food and Drug Administration–approved phase 1 clinical trial to test the safety of this antibody in humans.

The Next Steps: Taking Remyelination to Clinical Trial

The Right Patients at the Right Time

The next steps are equally critical. As Dr Weinshenker says, "To be successful, we need a great tool, and we need the right patients at the right time. The challenge is to find them." The right time is early in the disease when it is amenable to

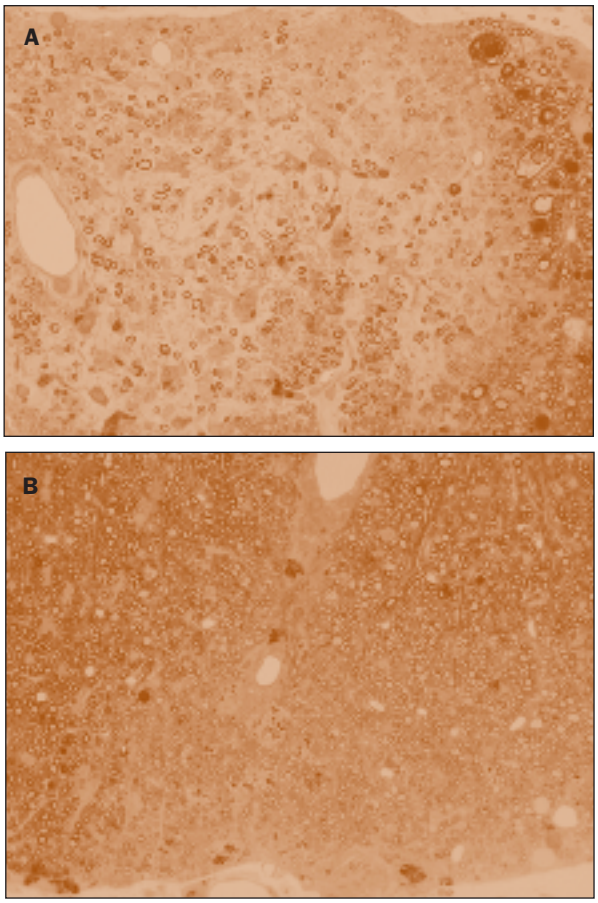

Figure 2. Photomicrograph of mouse spinal cord demonstrating limited repair in an untreated animal with demyelinating disease (A) and marked remyelination in an animal treated with a single 100-µg dose of the remyelination-promoting human IgM rHIgM22 (B).

treatment. The right patients would seem to be patients who are "good natural remyelinators" who fall into the 2 MS subgroups in which oligodendrocytes and/or progenitor cells are available to respond to IgM number 22. It is important to identify patients with the potential to repair using noninvasive tools. In the past year, the MS Lesion Project has found a strong correlation between evidence of remyelination on biopsy and a pattern of "ring enhancement" in MS lesions that shows up on MRI scans. This finding could greatly enlarge the pool of candidates for clinical trials.

Measuring Outcomes

- Is there a critical period for remyelination?
- Are factors affecting endogenous remyelination the same in the acute and chronic phases of the disease?
- How much myelin is needed for success?
- How many axons need to be repaired to prevent diffuse tissue damage?
- Can axon outgrowth be promoted through remyelination?

Answers to these questions rest on the ability to measure an outcome, which itself interacts with timing and patient selection. For example, in the short run, patients who are good remyelinators may remyelinate almost as well without treatment. The goal, however, is to prevent chronic degeneration, a process that may take years, so clinical signs must be combined with other objective measures of outcome. To monitor outcome, Mayo neuroradiologists, led by Bradley J. Erickson, MD, PhD, are developing ever more refined, innovative techniques such as identifying ring enhancement on MRI and increased levels of *N*-acetyl aspartic acid (NAA) by MR spectroscopy.

The Potential Impact of Remyelination

Multiple sclerosis affects 1 in 1,000 people in the Western world. Mayo Clinic Rochester sees more than 1,500 new cases of MS a year. Across its 3 campuses, Mayo Clinic has a team of specialists who have devoted their careers to the study and treatment of MS. Remyelination may be an important step in that direction. With a laboratory focused on the pathologic factors contributing to early and late disease, another invested in developing remyelination strategies, with a potentially effective antibody in production, and an imaging team devoted to finding a way to measure outcomes, Mayo Clinic is positioned to discover whether the promise of remyelination holds true.

Dr Weinshenker says, "If we are 80% successful in reducing or repairing lesions, we're going to have a very big impact, one that might very well prevent patients from progressive disease down the line." Dr Rodriguez anticipates that IgM antibody number 22, if successful, may be a major step toward therapeutic nervous system repair not only in patients with MS but also potentially for those with other degenerative CNS diseases and spinal cord injury. Aware of the challenges ahead, Dr Lucchinetti sees saving axons as one of the best means of preventing intractable disability in patients who "are getting worse, despite therapies that impact the inflammatory aspect of the disease."

Mayo Clinic Stroke Programs: Providing Acute Care in the Community and Beyond

The term "brain attack" has never really caught on in the public imagination. This is unfortunate because "stroke" often fails to invoke the sense of medical urgency, of life-and-death drama that "heart attack" does. Yet timely treatment for stroke is just as critical. Every minute counts. The initial symptoms of stroke—a slight tingling in the hand, a transient bout of slurred speech, for example—can be subtle and may not be recognized for what they are. And it is difficult for the public to comprehend the potentially devastating consequences of damage to the brain.

Even within the medical community, acute stroke may not translate into rapid intervention. For example, the administration of tissue plasminogen activator (t-PA) in the first 3 hours can limit the amount of brain tissue injured in an ischemic stroke. According to the American Stroke Association, of those eligible, an average of only 5% to 10% receive it. Administration of t-PA is one of 10 quality-of-care indicators Mayo Clinic Arizona tracks in compliance with the American Stroke Association's "Get With the Guidelines-Stroke" (GWTG-Stroke) program. For the past 3 years, 100% of their eligible patients received t-PA within the first 3 hours after onset.

Rapid Response: The Critical First Hour

There is a box, referred to as the "clot box," in the emergency department (ED) at Mayo Clinic Hospital in Phoenix, Arizona, that contains everything needed to administer intravenous t-PA. In that same ED is a large poster on the wall—a minute-by-minute timeline for acute stroke care. An ideal aspired to, it quickly became a reality as Mayo's stroke team shaved 20 minutes off the time from ED arrival to appropriate intervention (the "door to needle" response time). It required the formation and dedication of a rapid-response team, one that includes the very first persons

The stroke team, left to right: Timothy Ingall, MD, PhD, Bart M. Demaerschalk, MD, Maria I. Aguilar, MD, and David W. Dodick, MD

on the scene—the emergency medical services (EMS) responders. Neurologists and emergency physicians from Mayo Clinic Arizona helped the Phoenix and Scottsdale fire departments set up the Stroke Alert System and then trained their EMS personnel to evaluate stroke in the field and call in the diagnosis en route.

The minute the call comes in, a triage nurse at Mayo Clinic Hospital activates a single phone number that simultaneously pages all members of the stroke team, alerts radiology to prepare for an urgent CT scan, and notifies lab technicians and stroke team nurses. The patient must be seen within 10 minutes of arrival. The on-call stroke neurologist must be present within 20 minutes, the CT scan done within 25 minutes, and lab studies done and interpreted within 45 minutes. The goal is to deliver t-PA intravenously, if appropriate, within 60 minutes of the patient's arrival, a goal that has been met in 100% of cases since 2004. The same timeline applies for inpatients with acute stroke. Nurses and technicians throughout Mayo have been trained to recognize the symptoms and to call the coactivated page number.

Dedicated stroke care beyond the first hour includes delivery and monitoring of necessary medications (eg, lipid-lowering agents, antihypertensive treatments, antiplatelet medications, anticoagulants); providing speech, swallowing, physical, and occupational therapy as needed; and providing stroke education and counseling. Finally, it includes a follow-up visit with the neurologist within 4 to 6 weeks to help the patient sort through life changes, assess the need for continued services, and reiterate stroke prevention guidelines.

This level of dedication earned Mayo Clinic Arizona designation as a Phoenix Primary Stroke Center (PSC) in April 2003 and certification as a Joint Commission (JC) PSC in May 2006. With more than 85% compliance with GWTG-Stroke (Table), it was 1 of approximately 30 hospitals in the country awarded the American Stroke Association Annual Performance Achievement Award last year. St. Luke's Hospital, a Mayo Clinic facility in Jacksonville, is also a JC-certified PSC. Hospitals at Mayo Clinic Rochester are awaiting a JC site visit for final approval.

Mayo Clinic Arizona's Stroke Program

The stroke team, directed by Bart M. Demaerschalk, MD, includes neurologists Maria I. Aguilar, MD, David W. Dodick, MD, and Timothy Ingall, MD, PhD. One team member is on call 24 hours a day, 7 days a week. An endovascular surgical neuroradiologist, Brian W. Chong, MD, provides such treatments as mechanical clot retrieval and intra-arterial t-PA, offered also at Mayo Clinic's Jacksonville and Rochester sites and a select number of other facilities in the country.

Reflecting the complex nature of stroke, the stroke program at Mayo Clinic Arizona is guided by an interdisciplinary committee that includes members of the departments of neurology, neurosurgery, neuroradiology, vascular surgery, emergency medicine, internal medicine, critical care medicine, physical medicine and rehabilitation, and cardiology. Stroke patients are preferentially admitted to a specific ward with nurses who have expertise in neurologic and neurosurgical care and receive state-of-the art treatments such as intravenous thrombolytic therapy, intra-arterial thrombolysis, clot retrieval, stents, coils for aneurysms, and new drugs and interventions that are in clinical trials.

Reaching Out to the Community

Beginning in 1998, Dr Ingall and physicians and health care providers at other institutions helped establish the Phoenix Metropolitan Matrix of Primary Stroke Centers to ensure designated, hospital-based stroke care coverage in all areas of Phoenix. Bentley J. Bobrow, MD, a Mayo emergency medicine physician, and Dr Demaerschalk collaborated to develop ASPIRE, a program that tracks the response to and outcome of every patient with acute stroke called in by an EMS provider. Overall, these community efforts have resulted in a 20-fold increase in the timely delivery of t-PA to

Table. Standardized Stroke Measure Set Established by the Joint Commission*

- Deep vein thrombosis prophylaxis
- Discharged on antithrombotics
- Patients with atrial fibrillation receiving anticoagulation therapy
- Tissue plaminogen activator (t-PA) considered and administered
- Antithrombotic medication within 48 hours of hospitalization
- Lipid profile during hospitalization
- Screening for dysphagia
- Stroke education given
- Smoking cessation
- Plan for rehabilitation considered

* 80% compliance is required for designation as a Joint Commission–certified Primary Stroke Center, and 85% compliance is the award threshold for GWTG-Stroke.

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patients in the Phoenix area since 1998.

Stroke Telemedicine for Arizona Rural Residents (STARR) allows Mayo neurologists to provide diagnostic consultation to patients in the rural communities of Yuma and Kingman through a telemedicine program funded by the Arizona Department of Health Services. Via Internet access, Mayo physicians have site-independent, 2-way audio-video capability and can control a remote camera to conduct an examination and consultation with patients and ED physicians in rural emergency settings (Figure). CT scan images and lab reports can be reviewed, helping an on-site physician provide interventions such as t-PA in a timely manner. Patients who need services that are unavailable can be air-lifted to Mayo. "Before this program, this level of acute stroke care was in the exclusive domain of large metropolitan and academic centers. This is a community service that extends Mayo's care outside those areas," explains Dr Demaerschalk. "If it succeeds, it is hoped that the state will extend funding to new areas, improving stroke care throughout Arizona."

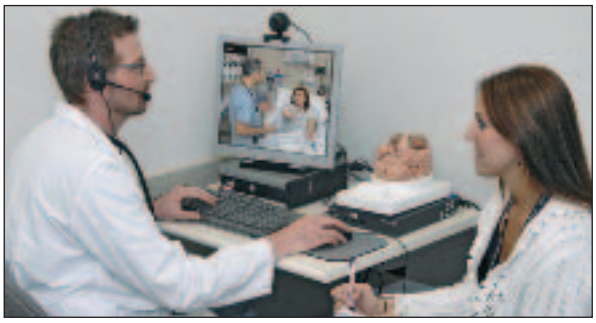

Figure. Vascular neurologist Bart M. Demaerschalk, MD, demonstrates how an urgent telemedicine consultation for an acute stroke patient is conducted, while clinical research coordinator Tiffany Koch collects and records the necessary data.

Expedited Patient Referrals to Mayo Clinic Departments of Neurology and Neurologic Surgery

While Mayo Clinic welcomes appointment requests for all neurologic and neurosurgical conditions, patients with the following conditions are offered expedited appointments:

1. **Cerebral aneurysms**
2. **Cerebral or spinal arteriovenous malformations**
3. **Brain, spinal cord, or peripheral nerve tumors**
4. **Epilepsy with indications for surgery**
5. **Carotid disease**

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200 First Street SW
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507-284-8008

Neurologic Consultation
507-284-1588

Non-Neurologic Consultation
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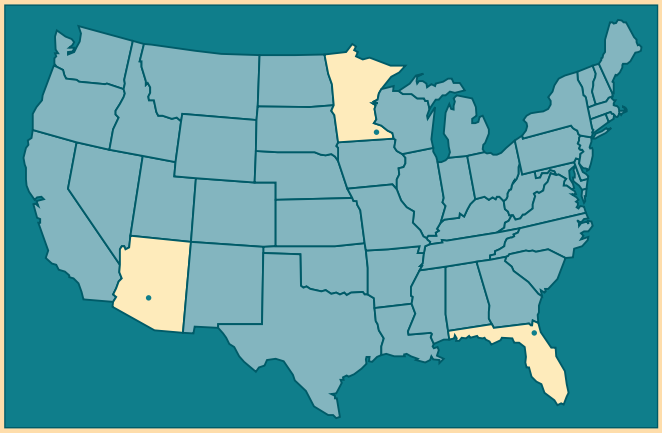

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